regulatory element. This provides further evidence for this previously proposed regulatory element downstream of *SHOX* and adds additional proof that increased dosage of this regulatory element region is a cause of short stature.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Calcitriol-Mediated Hypercalcemia in a Patient With Metastatic Gastrointestinal Stromal Tumor

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MON-LB69

Background: Hypercalcemia is a common complication of advanced malignancy, affecting up to 30% of cancer patients through various mechanisms (1). Hypercalcemia has rarely been described in gastrointestinal stromal tumors (GIST), with fewer than ten case reports as of 2018 (1,2). We describe a case of calcitriol-mediated hypercalcemia in a patient with GIST. Clinical Case: An 80-year-old woman with a history of metastatic GIST and nivolumab-induced type 1 diabetes and thyroiditis presented with dramatic progression of metastatic peritoneal disease and new severe hypercalcemia with acute kidney injury. On hospital admission, calcium (Ca) was 15.1 mg/dL (8.6-10.3 mg/dL), ionized Ca was 1.98 mmol/L (1.09-1.29 mmol/L), and creatinine was 2.56 mg/dL (0.6-1.3 mg/dL, baseline 1.8 mg/dL). She was treated with IV fluids and 45 mg of IV pamidronate with initial Ca improvement to 10.7 mg/dL over the next 48 hours. Additional workup showed that 25-hydroxyvitamin D was 18 ng/dL (20-50 ng/dL), PTH was 9 pg/mL (11-51 pg/mL), PTHrP was 3.1 pmol/L (0.0-3.4 pmol/L), and calcitriol was elevated to 172 pg/mL (19.9-79.3 pg/mL). Prior chest/abdomen/pelvis CT scans had not shown bony metastases or granulomas. After stopping IV fluids, Ca rose to 12.2 mg/dL the next day. Prednisone 20 mg daily was started which stabilized Ca levels and lowered calcitriol to 17.4 pg/mL after two weeks. She also began a new regimen of cabozantinib. Prednisone was tapered to 10 mg daily and she continues to maintain normal Ca levels with the addition of home health IV fluids three times a week. Conclusion: GIST tumors are a rare cause of hypercalcemia of malignancy. Although hypercalcemia of malignancy is most often due to tumor-secreted PTHrP or bony metastases, a small percentage of cases are mediated by excess calcitriol production. There is a growing number of case reports, including this case, to suggest that calcitriol-mediated hypercalcemia is the most common cause of hypercalcemia in GIST tumors (2-4). Glucocorticoids may be used to decrease calcitriol production and help maintain eucalcemia. Definitive therapy for hypercalcemia in these patients involves decreasing tumor burden by treatment of the underlying malignancy (3). References: (1) Stewart AF. Clinical practice. Hypercalcemia associated with cancer. N Engl J Med. 2005;352(4):373-9. (2) Hart T, Sinitsky D, Shamsiddinova A, Rohatgi A. Refractory hypercalcaemia secondary to localised gastrointestinal stromal tumour. Ann R Coll Surg Engl. 2018;100(6):e136-e138. (3) Hygum K, Wulff CN, Harsløf T, et al. Hypercalcemia in metastatic GIST caused by systemic elevated calcitriol: a case report and review of the literature. BMC Cancer. 2015;15:788. (4) Barbaryan A, Bailuc S, Poddutoori P, et al. Gastrointestinal Stromal Tumor Induced Hypercalcemia. Case Rep Oncol Med.2017;4972017.

Adrenal

ADRENAL CASE REPORTS II

A Case of BMAH With Aberrant Vasopressin Adrenal Receptors

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SUN-LB34

We report on a 42 - year old woman was referred following an incidental finding of bilateral macronodular (nodules > 1cm) adrenal glands after a computed tomography scan for investigation of an unrelated urological problem. Clear features of Cushing's syndrome were elicited on initial clincal evaluation and screening tests confirmed the diagnosis biochemically: midnight salivary cortisol 15.4nmol/L (0.2 - 3nmol/L), 24 hour urine free cortisol > 662.4nmol/L (8.3 - 118.7nmol/L per 24 hours) and an 8am serum cortisol measured 951nmol/L following 1mg dexamethasone suppression test (< 50nm/L). A serum adrenocorticotropic hormone (ACTH) measured 0.3pmol/L (1.6 - 13.9pmol/L) suggesting, in light of the CT findings, an adrenal source of the elevated cortisol. Screening for aberrant adrenal receptors to catecholamines, Gastric inhibitory peptide, ACTH, vasopressin and Gonadotropin releasing hormone was performed. A positive biochemical response to ACTH and vasopressin was noted as measured serum cortisol increased to 1600pmol/L (upper limit of the assay). Following a unilateral adrenalectomy, were pathological examination of the respected adrenal gland confirmed adrenal hyperplasia, the patient had both clinical and biochemical resolution of hypercortisolemia. 2 weeks following surgery her midnight salivary cortisol measured 2.1nmol/L (0.2 - 3nmol/L) and 24 hour urine free cortisol 218nmol/L (8.3 - 118.7nmol/L per 24 hours). Her 8am ACTH also increased to 2.9pmol/L (1.6 - 13.9pmol/L). Biochemical screening of her sister showed no evidence of Cushing's syndrome. Bilateral macronodualr hyperplasia (BMAH) represents a rare cause of Cushing's syndrome may be diagnosed incidentally and is often associated with an insidious onset. We use this case to highlight the complexities of provocative testing for aberrant adrenal receptors which occur with BMAH and the utilization of unilateral adrenalectomy to achieve clinical and biochemical remission of Cushing's syndrome thus, removing the need for post - operative steroid replacement following bilateral adrenalectomy.

Neuroendocrinology and Pituitary

PITUITARY TUMORS II

The Use of 11C Methionine PET CT In Localization of ACTH Dependent Cushing's Disease

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